Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: A staged approach A report from the International League Against Epilepsy Nonepileptic Seizures Task Force

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SUMMARY

An international consensus group of clinician-researchers in epilepsy, neurology, neuropsychology, and neuropsychiatry collaborated with the aim of developing clear guidance on standards for the diagnosis of psychogenic nonepileptic seizures (PNES). Because the gold standard of video electroencephalography (vEEG) is not available worldwide, or for every patient, the group delineated a staged approach to PNES diagnosis. Using a consensus review of the literature, this group evaluated key diagnostic approaches. These included: history, EEG, ambulatory EEG, vEEG/monitoring, neurophysiologic, neurohumoral, neuroimaging, neuropsychological testing, hypnosis, and conversation analysis. Levels of diagnostic certainty were developed including possible, probable, clinically established, and documented diagnosis, based on the availability of history, witnessed event, and investigations, including vEEG. The aim and hope of this report is to provide greater clarity about the process and certainty of the diagnosis of PNES, with the intent to improve the care for people with epilepsy and nonepileptic seizures.

KEY WORDS: Nonepileptic seizures, Epilepsy, Differential diagnosis, Electroencephalogram, Video EEG monitoring, Tests.

The International League Against Epilepsy (ILAE) has identified psychogenic nonepileptic seizures (PNES) as one of the 10 key neuropsychiatric issues associated with epilepsy (Kerr et al., 2011). The management of patients with PNES begins with an accurate diagnosis (LaFrance et al., 2013b). The misdiagnosis of PNES leads to inappropriate treatment

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Wiley Periodicals, Inc. © 2013 International League Against Epilepsy of presumed epilepsy, with significant risk of iatrogenic injury, morbidity, and cost to patients and to the health care system (Reuber et al., 2004a; LaFrance & Benbadis, 2006). Several studies have documented that misdiagnoses resulting from misinterpretations of the patients' history or of misreading electroencephalography (EEG) studies are common. The ILAE Commission on Neuropsychobiology Nonepileptic Seizures Task Force was charged with developing a consensus on minimal requirements for diagnosis of nonepileptic events. The ILAE Nonepileptic Seizures Task Force comprises an international group of epileptologists, neuropsychiatrists, and neuropsychologists. This article describes the development and content of the International Consensus Clinical Statement on PNES Diagnosis.

DEFINITIONS

Seizures can be divided into three major categories: epileptic seizures (ES), PNES, or physiologic nonepileptic events (NEEs) (Gates, 1998). Like epileptic seizures, PNES present as paroxysmal time-limited, alterations in motor, sensory, autonomic, and/or cognitive signs and symptoms, but unlike epilepsy, PNES are not caused by ictal epileptiform

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activity (LaFrance & Devinsky, 2004). In contrast to ES, which are a manifestation of excessive and hypersynchronous discharges in the brain, PNES have psychologic underpinnings and causes. Physiologic NEEs are neither epileptic nor psychogenic, rather they are events associated with systemic alterations that produce an ictus (e.g., convulsive syncope or hypoglycemic seizure).

PNES occur across cultures and continents. Events described as PNES and occurring in a similar context to PNES seen in industrially developed countries are reported from threshold or developing countries, as well. PNES therefore seem to represent a fairly universal human condition. The semiologies are described similarly across ethnicities and cultures (Yang et al., 1996; De Paola et al., 2006; Szabo et al., 2012).

The majority of patients with recurrent seizures are initially presumed to have epilepsy and are treated with antiepileptic drugs (AEDs) (Reuber et al., 2002a). When seizures continue, trials of AED polytherapy or referral for presurgical evaluation may follow. Because AEDs do not treat PNES and may exacerbate them (Niedermeyer et al., 1970), the early and accurate recognition of PNES, and their differentiation of ES and other paroxysmal disorders, is of paramount importance. The situation is complicated by the fact that epilepsy is a recognized risk factor for the development of PNES. The combination of ES and PNES occurs in 10% of patients with PNES (Benbadis et al., 2001), and this number may be higher, especially in more specialized settings (Reuber et al., 2003c).

In the differential diagnosis of seizures, the combination of video-EEG (vEEG) with the history of patients and witnesses offers a diagnostic "gold-standard" with high levels of certainty and excellent interrater reliability (IRR) (Syed et al., 2011). However, vEEG is not available in some locations, and in some patients, events cannot be recorded. Some providers have limited access even to routine epilepsy diagnostic equipment (Kvalsund & Birbeck, 2012). We, therefore, have addressed the problem of making a clinical diagnosis of PNES, either when vEEG is available or without vEEG data.

This report recognizes that, in practice, the diagnosis is often iterative, rather than a "one shot" process, and that the long-term clinical course may be important. The authors of this summary report also recognize that different levels of diagnostic certainty may be required for different scenarios (such as, diagnostic certainty levels may be different for research and for clinical purposes). Greater clarity about the process and certainty of the diagnosis of PNES will aid communication about this important public health problem and improve the care of patients with epilepsy and PNES.

Methods

An international group was identified comprising ILAE members who are clinician researchers and who regularly diagnose and treat patients with PNES, ensuring that they represented issues that were relevant for professionals working across the globe. Medline and PsycINFO database searches were undertaken for articles with keywords addressing seizure diagnosis, monitoring, video-EEG, nonepileptic seizures, hysteroepilepsy, pseudoseizures, and dissociative seizures.

Following the development of the report, the document was sent to current members of the Neuropsychobiology Commission of the ILAE, as well as the Therapeutics Commission of the ILAE for final review. The consensus concerning diagnosis of PNES is described below.

RESULTS

Features raising suspicion of PNES

Background factors

PNES tend to present in patients in their 20s and 30s, although the range includes children and the elderly (Lancman et al., 1994; Reuber et al., 2003c; Duncan et al., 2006). Three fourths of patients are women (Meierkord et al., 1991; Francis & Baker, 1999; Szaflarski et al., 2000; Reuber et al., 2003a). Approximately 10% of patients with PNES also have epilepsy (Lesser et al., 1983; Benbadis et al., 2001; Duncan & Oto, 2008b), but up to 30% of those with PNES who also have intellectual disability (ID) have additional ES (Duncan & Oto, 2008b). Up to 50% of patients with PNES report a precipitating event that might also be associated with epilepsy (Westbrook et al., 1998), and of those reporting head injury, almost 75% met criteria for mild traumatic brain injury (TBI) (LaFrance et al., 2013a). Approximately 70% of patients with PNES have other psychogenic disorders (King et al., 1982; Kloster, 1993; Ettinger et al., 1999; Reuber et al., 2007). Up to 70% of patients report antecedent trauma, which is of a sexual nature in up to 40% (Goodwin et al., 1979; Gross, 1979, 1986; Greig & Betts, 1992; Bowman, 1993; Sharpe & Faye, 2006). Current or previous mental health and psychosocial problems are common (Roy, 1979, 1980; Stewart et al., 1982; Wilkus et al., 1984; Lempert & Schmidt, 1990; Vein et al., 1994; Krishnamoorthy et al., 2001; Duncan & Oto, 2008a), though far from universal.

The pattern and triggering of events

Event frequency is higher in patients with PNES than those with epilepsy (Jedrzejczak et al., 1999). Recurrent hospital admissions with apparent seizure status or daily convulsive events suggest PNES (Reuber et al., 2003b), especially when reported by a well and alert patient.

The triggering of events by stressful or difficult situations should suggest PNES, although only a minority of patients report it at initial clinical assessment (Duncan & Oto, 2008a). Patients with ID appear to have situational triggers more commonly (Duncan & Oto, 2008b), and some patients tend to have PNES in medical situations (McGonigal et al., 2002), such as in scanners and during consultations. A variety of physical triggers not usually associated with ES or syncope, such as change in lighting conditions and physical activities, may be reported. Photic stimulation and hyperventilation commonly provoke PNES during EEG recording (Leis et al., 1992), and they may arise on recovery from a general anesthetic (Reuber et al., 2000; Lichter et al., 2004).

PNES as a disorder may be triggered by surgery (including epilepsy surgery) and physical trauma (Glosser et al., 1999; Reuber et al., 2002d; Duncan & Oto, 2008a). The description or observation of a partial or transient response to antiepileptic drugs occurs in approximately 40% (Oto et al., 2005).

Background factors may distinguish populations of patients with PNES or epilepsy, but are of limited diagnostic use on an individual level. Furthermore, some factors (such as antecedent sexual abuse) may not become known until after the diagnosis has been made.

The clinical semiology of the events

Individual elements of seizure semiology (Reuber & Elger, 2003; Devinsky et al., 2011) are unreliable as diagnostic discriminators (Syed et al., 2011). Tables listing semiologic elements and comparing their frequency in ES versus PNES have been published. Such tables can be useful to summarize the literature, but are of limited clinical use, largely because comparisons published to date ignore important semiologic subdivisions. For example, in reality, the differential diagnosis of convulsive PNES is with tonic– clonic ES, whereas the differential diagnosis of a "swoon" type PNES is with vasovagal or cardiac syncope. As an example, a table from Avbersek and Sisodiya (2010) is reproduced, which does take some semiologic subdivisions into account (See Table 1).

The second issue is that the tables that have been published do not always distinguish whether the described features were recorded on the basis of eyewitness report or the examination of video footage, and there is good evidence that report and event semiologies are not the same (Sved et al., 2011). For example, the table of Avbersek and Sisodiya (2010) cites "occurrence from sleep" as having 100% specificity for ES. This is correct only for seizures from EEG confirmed sleep (for caveat, see Orbach et al., 2003). However, at the time of initial clinical assessment, approximately half of patients with PNES give a history of events "arising from sleep" (Duncan et al., 2004). Reported seizures from sleep should therefore not be taken as good evidence for epilepsy, except if the events occur only during sleep (Duncan et al., 2004). In this instance, applying insights based on vEEG recordings to data obtained by taking a history during an initial clinical assessment is likely to mislead.

Clusters of semiologic elements may differentiate PNES more clearly from ES (Hubsch et al., 2011; Reuber & Duncan, 2011; Syed et al., 2011), but it remains unclear whether this approach can be applied in the clinical setting. The most common clinical patterns of PNES observed include "convulsive" or "thrashing," where patients have loss of responsiveness with variable movements of limbs, head, and trunk (usually fine or coarse tremors); and "swoon," "catatonic," or "pseudosyncope," where patients fall down and lie still, with eyes closed and unresponsive (Gumnit & Gates, 1986; Gates et al., 1991; Lancman et al., 1994). A significant minority of patients have "dialeptic" or "absence"-like events (Seneviratne et al., 2010; Szabo et al., 2012), with the predominant symptomatology consisting of alteration in consciousness (Lüders et al., 1998).

Duration of PNES is longer than ES (Jedrzejczak et al., 1999; Selwa et al., 2000). Convulsive seizures, the motor features of which habitually last longer than 2 min, should be examined for possibility of PNES (Selwa et al., 2000), and a duration of >10 min strongly suggests PNES (Dworetzky et al., 2006). Out of phase limb movements and side-to-side head movements, especially with coordinated alternating agonist and antagonist activity (i.e., tremor), are highly suggestive of PNES (Gates et al., 1991; Leis et al., 1992; Selwa et al., 2000). Thrashing movements may be less useful, as they are seen in some types of frontal ES (Kanner et al., 1990; Saygi et al., 1992), albeit there do seem to be useful clinical discriminators in that event. In particular, short duration, tonic posturing, and seizures occurring during sleep only would favor a diagnosis of frontal lobe epilepsy (FLE) (Kanner et al., 1990). Forward pelvic thrusting is probably relatively uncommon in both epilepsy and PNES and is of limited discriminating value (Gates et al., 1985; Saygi et al., 1992; Geyer et al., 2000). Generalized tonicclonic (GTC) ES motor activity frequency declines gradually over the course of the ictus while amplitude increases. In PNES, the frequency remains unchanged throughout while amplitude is variable (Vinton et al., 2004).

"Swoon" type events should raise suspicion of PNES if prolonged over a minute (Gates et al., 1991; Jedrzejczak et al., 1999). This presentation is not a usual manifestation of epilepsy, but may result from vasovagal or cardiac syncope. Atonic ES are much shorter and typically occur in the epilepsies with other seizure types, for example, Lennox-Gastaut syndrome. Two important caveats apply. If acquiring a description, the doctor must be clear that the witness has seen the onset of the episode: Patients may well lie still for a time in the postictal phase of a tonic–clonic seizure. If witnessing this type of event, the doctor should make an immediate clinical check for a cardiac output. If the pulse is strong, regular, and of a reasonable rate, then checks for responsiveness, resistance to eye opening, self-protective maneuvers, and so on, are indicated.

Vocalization in PNES occurs during or after seizures, and may be complex, with affective content, whereas in ES it occurs at the beginning, is primitive, and has no emotional expression (Gulick et al., 1982; Luther et al., 1982; Gates

Signs that favor PNES	Evidence from primary studies	Sensitivity (%) for PNES	Specificity (%) for PNES
Long duration	Good	_	_
Fluctuating course	Good	69 (events)	96
Asynchronous movements	Good (frontal lobe partial seizures excluded)	47–88 (patients)	96-100
		44–96 (events)	93–96
		9–56 (patients)	93-100
Pelvic thrusting	Good (frontal lobe partial seizures excluded)	I–31 (events)	96-100
		7.4-44 (patients)	92-100
Side to side head or body movement	Good (convulsive events only)	25–63 (events)	96-100
	· · ·	I 5–36 (patients)	92-100
Closed eyes	Good	34–88 (events)	74–100
		52–96 (patients)	97
lctal crying	Good	I3–I4 (events)	100
		3.7–37 (patients)	100
Memory recall	Good	63 (events)	96
		77–88 (patients)	90
Signs that favor ES	Evidence from primary studies	Sensitivity for ES	Specificity for ES
Occurrence from EEG-confirmed sleep	Good	31–59 (events)	100
Postictal confusion	Good	_ 61–100 (events)	88
		67 (patients)	84
Stertorous breathing	Good (convulsive events only)	61–91 (events)	100
Other signs	Evidence from primary studies	_	_
Gradual onset	Insufficient		
Nonstereotyped events	Insufficient		
Flailing or thrashing movements	Insufficient		
Opisthotonus "arc en cercle"	Insufficient		
Tongue biting	Insufficient		
Urinary incontinence	Insufficient		

et al., 1985; Kanner et al., 1990; Leis et al., 1992; Saygi et al., 1992). Signs of emotional distress suggest PNES (Luther et al., 1982; Bergen & Ristanovic, 1993; Walczak & Bogolioubov, 1996). Histories of urinary incontinence and injury (Luther et al., 1982; Wilkus et al., 1984; Gumnit & Gates, 1986; Meierkord et al., 1991; Peguero et al., 1995; Reuber et al., 2003a) are poor discriminators, and PNES are just as likely to be stereotyped as ES (Gulick et al., 1982; Devinsky et al., 1996).

In summary, semiologic differences and overlaps between ES and PNES exist, and illustrate the adage, "one sign or symptom does not a diagnosis make." The diagnosis of PNES requires neurologic (semiology and EEG) and psychiatric (psychosocial history and diagnostic criteria) internal consistency, as discussed below (Table 1).

Psychiatric diagnosis (DSM-IV-TR/DSM-5 and ICD-10 criteria)

The great majority of PNES are classified as mental disorders in the current medical nosologies (only malingered seizures are not considered a mental disorder). However, these nosologies, including the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR), are of limited use in clinical practice. In effect, the DSM-IV-TR proposed a two-stage diagnostic process. In the first stage, an explanatory medical cause for the symptoms (seizures) needs to be excluded. In the second stage, the DSM criteria are used to allocate the medically unexplained seizures to conversion disorder or, in some cases, to dissociative or anxiety disorders. Some nosologic subjectivity exists in that a diagnosis of Panic Disorder with comorbid Conversion Seizures may relate to both, for example, patients whose PNES arise out of typical panic symptoms and patients with two different types of events. The Conversion Disorder criteria in DSM-5 have been changed by guiding users to make a positive diagnosis based on symptom presentation and by relegating a psychological stressor from a criterion to a note (Stone et al., 2011), aligning the DSM system with ICD-10. However, the classification of PNES as Conversion Seizures in DSM-5 remains the same as in the previous DSM version.

Confirming the diagnosis of PNES

The diagnosis of PNES is best confirmed by recording events simultaneously on video and EEG, finding an absence of ictal EEG changes (and the presence of normal awake EEG rhythms) before, during, and after the event. If this is done, a positive diagnosis of the event recorded can be made in the great majority of cases. However, the absence of EEG change in and of itself is not always diagnostic, and it remains important that a vEEG diagnosis with PNES-consistent semiology is made in the context of clinical data. Events that do not approximately correspond with known PNES semiology should be examined critically, even if not accompanied by EEG change. If the recorded events are clinically compatible with simple partial ES (i.e., consist of very localized motor movements, or a subjective experience only; Kanner et al., 1990), or if they are clinically compatible with hypermotor FLE, (Saygi et al., 1992), then the absence of EEG change does not necessarily indicate PNES (although clinical characteristics and electrocardiography [ECG] changes may—see above).

To state the obvious, for confirmation of normal rhythms throughout an event, the EEG has to be visible, and not completely obscured by muscle artifact. Crucially, any recorded event must be confirmed by an eyewitness as typical of those occurring in daily life. If clinical descriptions suggest more than one event type, then an occurrence of each type must, whenever possible, be recorded.

The concurrent recording of ECG during vEEG is essential. Ictal heart rate is higher and the ictal heart rate increase is more rapid in epilepsy than in PNES (Donati et al., 1996; Opherk & Hirsch, 2002; de Oliveira et al., 2007). Unlike that seen in ES, the heart rate increase in PNES is usually commensurate with the physical activity involved in the seizure (Reinsberger et al., 2012).

The majority of patients with PNES will produce an event within the first few hours of vEEG recording (Ettinger et al., 1999). The use of suggestion techniques ranging from simple verbal suggestion to injection of saline may improve rate of seizure capture (McGonigal et al., 2002, 2004; Benbadis et al., 2004; Varela et al., 2007), and may allow avoidance of a longer admission to hospital in as many as half of patients (McGonigal et al., 2004). Ethical concerns are raised with saline injections and placebo swipes (Stagno & Smith, 1997); however, these concerns should not be an issue for diagnosis if routine activation procedures (hyperventilation and photic stimulation) are used (Benbadis et al., 2000). Some authors support use of simple suggestion techniques if the patient is clearly informed of what is being done and why (this does not seem to prevent patients from having events during recording, McGonigal et al., 2002).

Some patients do not have seizures in an observed setting. Outpatient ambulatory EEG may be useful in this circumstance, particularly if there is a caregiver who can give good descriptions of the events that have been recorded, or can provide video recordings of them. Without such clinical information, ambulatory EEG has to be interpreted with caution. In conjunction with clear clinical data, video alone can allow a reasonably confident diagnosis in some cases, and seems to be most accurate in seizures where there is motor activity (King et al., 1982; Chen et al., 2008). However, it seldom captures the beginning of the event, and this has to be borne in mind when interpreting the recording: Behavioral disturbances in the postictal phase of ES may superficially resemble PNES.

Diagnostic supplements

Only ictal EEG can be used to differentiate PNES from ES definitively at the individual level. However, a number of neurophysiologic, neurohumoral, and neuropsychological tests allow discrimination at the population level. After conducting a thorough history, mental status, and neurologic examination, these instruments can be used to assist in the diagnosis of PNES, and are summarized below.

Physiologic measures

Serologic measures have been used in differentiating epilepsy from PNES, the most useful being prolactin (PRL). Elevated serum PRL in patients with GTC ES helped distinguish epilepsy from PNES (Trimble, 1978). Many studies have since been conducted measuring PRL in PNES, finding that the absence of postictal PRL rise predicts PNES with a mean sensitivity to PNES of 89% across the studies (Cragar et al., 2002). Furthermore, studies have shown that serum PRL levels are elevated on average in 88% of cases of GTC ES, in 64% of temporal lobe complex partial (CPS) ES, and in 12% of simple partial ES. Reasons for "false positive" PRL tests include treatment with dopamine antagonists and some tricvclic antidepressants, breast stimulation, and svncope. "False negatives" occur with use of a dopamine agonist, or with status epilepticus, because PRL has a short half-life and may attenuate in postictal release (Bauer, 1996). PRL may also fail to rise after frontal lobe ES. The American Academy of Neurology Therapeutics and Technology Assessment Subcommittee concluded that a twice normal relative or absolute serum PRL rise, from blood drawn 10-20 min after the onset of the ictus, compared against a baseline nonictal PRL, is a useful adjunct in the differentiation of GTC ES or CPS ES from PNES (Chen et al., 2005).

Investigations of serum cortisol and the dexamethasone suppression test (DST) have not reliably differentiated PNES, depression, or epilepsy groups (Tunca et al., 1996, 2000). Bakvis has shown that a history of sexual abuse in conversion disorder subjects is associated with greater baseline cortisol levels (Bakvis et al., 2010). Bakvis demonstrated that patients with PNES have increased basal diurnal cortisol levels associated with a history of sexual trauma (Bakvis et al., 2010) and lower heart rate variability at baseline, suggesting greater sympathetic activity (Bakvis et al., 2009). There were no differences in the DST or salivary amylase measures.

Other serum measure studies to differentiate GTC ES from PNES have included the use of elevations in peripheral white blood count (Shah et al., 2001), cortisol (Pritchard et al., 1985), creatine kinase (Wyllie et al., 1985), and neuron-specific enolase (Rabinowicz et al., 1996); however, the limited discriminative power of these serologic tests in differentiating ES from PNES has been discussed (Willert et al., 2004). Capillary oxygen saturation on pulse oximetry is lower for epilepsy than for PNES (James et al., 1991). Brain derived neurotrophic factor (BDNF) levels have been shown to be lower in patients with PNES than healthy controls, but did not differ from patients with epilepsy (LaFrance et al., 2010b).

Neuroimaging

Most patients with epilepsy have normal magnetic resonance imaging (MRI) studies (Reuber et al., 2003c), and a significant number of patients with lone PNES have abnormalities (Reuber et al., 2002c; LaFrance et al., 2009, 2010a). More recently, structural and functional imaging studies in patients with PNES have documented changes in cortical and cerebellar regions at group level (Labate et al., 2012); in functional connectivity between emotional, cognitive, and motor regions (van der Kruijs et al., 2012b); and between structural and functional connectivity network coupling (Ding et al., 2013). More studies are needed to determine if there are actual conversion/dissociation networks (van der Kruijs et al., 2012a).

Implications for clinical practice. Neuroimaging findings are of modest differential diagnostic value at present. Lesions with epileptogenic potential (such as mesial temporal sclerosis) are more commonly found in patients with epilepsy, but have also been described in patients with PNES and are clearly not sufficient for a diagnosis of epilepsy. Most patients first presenting with epilepsy have normal MRI scans (Kotsopoulos et al., 2003).

Neuropsychological testing

Neuropsychological testing can assess cognitive, emotional, personality, and effort measures. In terms of the neuropsychological profile of patient with PNES and the ability of such profiles to distinguish patients with PNES from those with epilepsy, data are varied. Neuropsychological tests do not distinguish ES from PNES at the individual level. The Table S1 presents, in reverse chronological order, a summary of the major studies to date for these areas, but it is not meant to be exhaustive. Clinical applications are summarized below.

Cognitive factors

There is some evidence that implies that patients with PNES have generally higher IQs than those with ES (Drake et al., 1993), but also that there are no differences between PNES and epilepsy groups (Dodrill, 2008). Other publications suggest that PNES are associated with neuropsychological impairment in a number of cognitive domains (Kalogjera-Sackellares & Sackellares, 1999).

Implications of cognitive measures for clinical practice. Although cognitive assessments of patients with PNES are often undertaken in clinical settings, their differential diagnostic value is questionable (Beghi et al., 2006). These in-depth assessments do, however, provide a neuropsychological profile of PNES patients, highlighting the specific cognitive difficulties encountered by this patient group, offering a comparison to the cognitive impairments found in patients with epilepsy, and contributing to the theoretical explanation as to why patients with PNES may present with such neuropsychological abnormalities. Observed differences between self-perception of cognitive functioning and objective performance have highlighted the need for such comprehensive assessments when evaluating neuropsychological functioning, as opposed to relying on self report (Breier et al., 1998; Fargo et al., 2004; Prigatano & Kirlin, 2009).

Emotional factors

Many studies have examined the emotional factors associated with PNES, such as comorbid psychiatric disorders and measures of anxiety, depression, and quality of life.

Implications of emotional measures for clinical practice. The approach to investigating the comorbidities has been varied in the methodology employed, including applied measures, sample sizes, and the absence of controls or patients with epilepsy. The emotional neuropsychological literature, therefore, provides data about emotional characteristics that are essentially descriptive of the population with PNES, rather than providing direct support for the diagnosis of PNES. However, the nature of the findings may be useful with respect to treatment formulations and the planning of interventions.

Personality measures

It is widely reported that patients with PNES may show personality disorders. Apart from interview methods like the Structured Clinical Interview for DSM Diagnosis (SCID) or Mini-International Neuropsychiatric Interview (MINI) to diagnose Axis I or II diagnoses, studies have employed self-report measures such as the Minnesota Multiphasic Personality Inventory (MMPI) or the MMPI-2 to compare patients with PNES to those with epilepsy. Studies of mixed quality have suggested that personality characteristics may differ between patients with only PNES and patients with both PNES and ES (Kuyk et al., 2003). However, reports (Baillès et al., 2004), for example, indicating the presence of multiple MMPI scale elevations, the absence of a single personality profile, the characterization of different personality profiles dependent on PNES semiology (e.g., Griffith et al., 2007), or the presence of a history of childhood trauma (e.g., Pintor et al., 2002) make diagnostic use of personality scales more difficult to establish a "PNES personality". Considering personality traits rather than disorders may be helpful (Reuber et al., 2004b).

Implications of personality measures for clinical practice. Despite the considerable interest in the use of personality measures with patients with PNES, most studies have not reported sensitivity and specificity data. Indeed (Cuthill & Espie, 2005) suggest that personality findings rarely indicate high sensitivity and specificity—the findings being generally better at excluding a possible diagnosis. It also has been suggested that MMPI profiles may be complex and cannot be seen as reflecting a unifying psychological mechanism (Vanderzant et al., 1986; Kalogjera-Sackellares & Sackellares, 1997). It is unlikely that the use of personality profiles can contribute any diagnostic certainty when evaluating patients for PNES but can still contribute to the characterization of the individual's presentation and therefore help shape their subsequent therapy.

Effort testing

A number of studies looking at malingering and underperformance in patients presenting with PNES have addressed the concept of exaggeration of cognitive symptoms or poor effort on cognitive testing (Loring et al., 2005; Drane et al., 2006; Locke et al., 2006; Dodrill, 2008). In general it has been concluded that more patients with PNES than epilepsy fail tests of effort and that cognitive impairment reported by patients with PNES appears to be more a function of motivational factors rather than verifiable neuropathology, although this may not necessarily represent *intentional* malingering; however, there is also evidence that patients with ES may not exert maximal effort on tests. For both ES and PNES, performance on effort tests predicts performance on formal cognitive testing (Locke et al., 2006).

Implications of effort measures for clinical practice. Despite the limited evidence, a number of reports have stressed the importance of differentiating between PNES and malingering due to the implications for misdiagnosis and treatment (Savard et al., 1988; Bhatia, 2004). However, at present there is no convincing evidence that the use of tests of effort (also referred to as symptom validity tests) will enhance the diagnostic process for PNES as compared to epilepsy.

Other diagnostic techniques

Hypnosis

The recent use of hypnosis in the diagnostic process of PNES includes its use in seizure provocation, which was tested in samples in the recent literature. Although events provoked by hypnosis are more likely to be PNES than epileptic seizures, the sensitivity and specificity has varied (sensitivity 46–77%; specificity 88–95%; Barry et al., 2000; Khan et al., 2009). Other approaches have used hypnosis to reverse amnesia for the seizure itself, which would not be expected after an epileptic seizure; Kuyk et al. (1999) demonstrated that the hypnotic recall technique yielded a specificity of 100% and sensitivity of 85% for PNES diagnoses. Studies have also indicated that patients with PNES score more highly than patients with epilepsy on measures of hypnotizability such as the Hypnotic Induction Profile (Kuyk et al., 1999; Barry et al., 2000; Khan et al., 2009), and these measures may be a useful adjunct, although their own sensitivity/specificity in the diagnostic procedure is less clear.

Implications for clinical practice. Hypnotic techniques have been reported as diagnostically (and therapeutically) useful by highly trained individuals with extensive experience with PNES. It is unclear at present how well readily these techniques could be applied more widely or how accurate diagnoses based on hypnotic techniques would be if the procedure was carried out by less experienced practitioners.

Conversation analysis (CA)

Although the process of taking the history from the patient and (ideally) a seizure witness remains the cornerstone of the diagnosis of PNES, little is known about the sensitivity, specificity, and IRR of "history-taking." There is emerging evidence of the diagnostic value of questions about clusters of semiologic elements on self-report questionnaires; however, the differentiating potential of such questions posed during the process of history-taking in the outpatient clinic has not been formally examined. Studies demonstrate that how patients share their subjective seizure experience with the doctor can help with the differential diagnosis of epilepsy and PNES (Schwabe et al., 2008; Plug & Reuber, 2009; Plug et al., 2009a; Reuber et al., 2009). Whereas patients with epilepsy readily focus on subjective seizure symptoms and tend to give detailed accounts of these symptoms characterized by extensive formulation effort (including reformulations, re-starts, neologisms, pauses), patients with PNES tend to focus on the situations in which seizures have occurred or the consequences of their seizures. Subjective seizure symptoms may be listed but are not described in detail. When the doctor tries to direct the patient's attention to particularly memorable seizures (e.g., the first, last, or worst seizure), patients with PNES commonly show focusing resistance, for instance by not providing further information or by generalizing rapidly to the description of their events in general. In contrast, patients with epilepsy readily provide more information about their subjective seizure symptoms in these particular seizures. Significant differences in preferred metaphoric conceptualizations are present. Whereas patients with epilepsy tended to describe their seizures as acting

independently (and often as doing something to the patient), those with PNES preferred metaphors depicting their seizures as a place or space they traveled through or to which they were confined (Plug et al., 2009b). Patients with PNES catastrophize their seizure experiences compared to patients with ES, who tend to normalize their seizure experiences when talking to a doctor (Robson et al., 2012).

Implications for clinical practice. These studies have demonstrated that the observation of patients' communication behavior in the interaction with neurologists has differential diagnostic potential. However, most of this work has been based on the detailed post hoc transcript-based analysis of conversations with patients with chronic seizure disorders. It remains to be shown which features can help with the differential diagnosis of patients who present with first seizures, and which (if any) of the described diagnostic features clinicians can identify "online," as they are talking to patients.

In summary, regarding adjunctive measures, we suggest that identification of multiple background factors should raise the clinical index of suspicion that the patient has PNES, but is not diagnostic. Data related to the seizures themselves can be more helpful for the diagnosis of PNES, and data related to personality/neuropsychological deficits may aid in the determination of underlying causes and best possible treatment.

Diagnosis of PNES—levels of certainty

Any diagnosis is associated with a particular level of uncertainty. Even the EEG recording of typical events

occasionally gives rise to a wrong diagnosis. This may be because ictal EEG changes obscured by muscle artifact have been incautiously labeled as normal, or because vEEG findings are assessed without taking account of their clinical context. As stated earlier, some types of epileptic seizures are usually not accompanied by ictal EEG changes. Taking this into account, and in the hands of experienced operators, vEEG confirms the nature of the recorded episode with a high degree of diagnostic certainty (Syed et al., 2011). Thorough clinical history elicits whether there is likely to be one or more habitual event types. The video allows comparison of the recorded events with those previously reported to ensure they are the same and that all event types have been captured.

The diagnosis of PNES may be based on different combinations of data. The combination of patient history, witness reports, clinician observations, and ictal and interictal EEG and ictal video is used for the diagnostic determination. We suggest four categories of certainty, based on common scenarios and the combination of the data, reflective of clinical practice. Each level increases with the combination of witness and EEG. It is not possible to devise categories based on all possible combinations of clinical data. One important caveat is that a normal interictal EEG does not exclude epilepsy or confirm PNES. Similarly, an abnormal interictal EEG does not confirm epilepsy or exclude PNES. This is discussed at length, below. Another caveat is that patients may have mixed epilepsy and PNES, and therefore, different seizure semiologies must be evaluated and classified independently (Table 2).

	History	Witnessed event	EEG
Diagnostic Level			
Possible	+	By witness or self-report/description	No epileptiform activity in routine or sleep-deprived interictal EEG
Probable	+	By clinician who reviewed video recording or in person, showing semiology typical of PNES	No epileptiform activity in routine or sleep-deprived <i>interictal</i> EEG
Clinically established	+	By clinician experienced in diagnosis of seizure disorders (on video or in person), showing semiology typical of PNES, while not on EEG	No epileptiform activity in routine or ambulatory <i>ictal</i> EEG during a typical ictus/event in which the semiology would make ictal epileptiform EEG activity expectable during equivalent epileptic seizures
Documented	+	By clinician experienced in diagnosis of seizure disorders, showing semiology typical of PNES, while on video EEG	No epileptiform activity immediately before, during or after ictus captured on <i>ictal</i> video EEG with typical PNES semiology

e maneuvers or forced eye closure during unresponsiveness or normal postictal prolactin levels? igi

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Possible PNES (e.g., based on clinical history from patient/ witness(es) and normal interictal EEG). A basic minimum diagnostic data set should be available in the great majority of clinical and service situations. This would consist of the patient's history and description of events, and if at all possible, an eyewitness description of events. These should indicate a specific event (or specific types of events, if more than one semiology exists) clinically typical of PNES. A number of studies have identified factual items in the history as well as linguistic and interactional features that distinguish between patients describing PNES and those describing epileptic seizures (see Conversation analysis section, above). Certain behaviors are not confirmatory of PNES. For example, seizures occurring in the presence of environmental or emotional stressors are not strong indicators of PNES or rule out epilepsy (Haut et al., 2003). In addition, seizures stopped by behavioral techniques are not strongly confirmatory of PNES (Reiter & Andrews, 2000). Obviously, a history and witnesses' description suggestive of PNES and an abnormal interictal EEG could be consistent with a diagnosis of possible PNES; however, in the absence of a clinician observing the ictus on video or in person, and with the question of interictal epileptiform discharges (IEDs), an alternative diagnosis of epilepsy would have to be considered very carefully.

Probable PNES (e.g., clinical history, clinician review of video recording of events or in person and normal interictal EEG). The reports of patients and witnesses may not match the actual semiology of observed seizures (Syed et al., 2011). Increasingly, home video recording is available to caregivers and relatives and can help to convey a clearer picture of the seizures. Although it may not be possible to distinguish clonic movements, tremors, or thrashing movements on the basis of an eyewitness account, the examination of events on a video recording usually allows experts to distinguish these events (Chen et al., 2008). One caveat is that some phone cameras have a low frame rate, which can make smooth physiologic movements look jerky and epileptic. Apart from the absence of EEG recording, the major disadvantage of home video is that the recording seldom captures the beginning of the event. It is important to note that the postictal phase of some epileptic seizures may look like PNES, and this can mislead. However, overall, video footage that can be reviewed by a clinician is likely to add to the certainty of a clinical diagnosis. Not being able to observe the onset of the seizure or a clinician who lacks experience in ictal assessment of patients would make PNES "probable." If IEDs are present on interictal EEG, in the absence of a video paired with EEG, a diagnosis of epilepsy must have been made unlikely, for instance, before making a probable PNES diagnosis. A normal interictal EEG and witnessing a non-vEEG seizure consistent with PNES characterize the diagnostic level of probable PNES.

Clinically established PNES (e.g., clinical history, clinician witness, plus ambulatory EEG recording of habitual event(s) without video). Generally, ambulatory EEG without video recording is more widely available than vEEG and has been used to differentiate epilepsy from PNES (Binnie, 1987). It has the advantage of allowing the patient to carry on a closer approximation to normal life during recording, but the disadvantage of not providing a timelocked recorded clinical correlate for the EEG (ambulatory EEG with video is available in some areas, and this is discussed later). Its success depends crucially on having good quality descriptions of habitual event(s), and a clear indication that any recorded events conform to that description. The event described should be clinically incompatible with simple partial seizures (whether small motor seizures, or experiential seizures) or hypermotor frontal lobe seizures in which ictal EEG changes may be lacking. The diagnostic yield of ambulatory EEG may be increased if it is combined with home video recording if video monitoring is not available with the monitoring equipment.

A diagnosis of clinically established PNES would also be considered if a clinician witness observed a seizure and documented examination findings typically found in PNES (for instance resisted eye-opening, degree of interaction with partial responsiveness during the seizure, or cessation of seizure activity with "talking down" by the physician). The label clinically established PNES would also be appropriate if a clinician was able to review a non-EEG event (by video or in person), and independently, an ictal non-vEEG capturing a typical event, showing no epileptiform activity immediately before, during, or after the ictus. Note that interictal abnormalities may be present.

Documented PNES (e.g., clinical history plus vEEG recording of habitual events). As an adjunct to good clinical data, the vEEG recording of habitual events provides the most reliable diagnosis of PNES. The procedure begins with a detailed recording of seizure description from patients and witnesses. Classically, the patient is admitted to the monitoring unit for a period of days, and recorded continuously during that time. Video footage can be examined to ensure that the recorded event is clinically a PNES, is clinically incompatible with a simple partial epileptic seizure, a hypermotor frontal lobe epileptic seizure, or a GTC epileptic seizure, and that the simultaneous EEG shows the patient's normal awake trace before, during, and after the event. While the use of provocation techniques (e.g., suggestion or placebo) has been discouraged more recently (Gates, 2001) because of the potential to compromise alliance (Stagno & Smith, 1997), routine EEG-activating procedures by sensory stimulation (e.g., photic-stimulation and hyperventilation) are not deceptive and allow clinicians to capture typical PNES in about two thirds of patients during brief vEEG recordings not necessitating admission to hospital (McGonigal et al., 2002). Documented PNES would,

therefore, require the ictus captured on vEEG to be reviewed by the clinician, pairing the behavior with the electrophysiology. Confirming that the recorded event(s) are typical of the patient's habitual seizures is necessary to establish documented PNES.

Other diagnostic indicators for consideration: ruling out mixed epilepsy with PNES. The diagnosis of epilepsy is also a clinical diagnosis. A detailed, accurate history is a fundamental practice in making the diagnosis. Clinical and laboratory criteria supporting a diagnosis of epilepsy are reviewed elsewhere (Drazkowski & Chung, 2010) and should be applied when the combination of PNES and epilepsy is being considered.

Interictal EEG findings in the healthy and in PNES. Interictal EEG is widely available worldwide, and most patients in whom the diagnosis of PNES is suspected will have had at least one routine EEG recording. Interictal EEG data should be interpreted with great care. It is not capable of making or excluding a diagnosis of PNES, nor is it capable of excluding epilepsy, as its false-negative rate is significant. Epileptiform abnormalities occur in only about 2% of healthy individuals or patients with PNES (and no additional epileptic seizures; Kotsopoulos et al., 2003), so specific EEG abnormalities have greater diagnostic value and may indicate a likelihood that the patient is likely to have spontaneous epileptic seizures. Nonspecific EEG abnormalities are common in PNES and epilepsy (as well as in patients who have syncopal episodes; Reuber et al., 2002b). A number of articles describe the risk of misinterpretation of EEG rhythms as epileptiform (Benbadis & Tatum, 2003; Benbadis. 2007). One study demonstrates that the overinterpretation of nonspecific EEG changes was one of the most common reasons that 25% of patients referred to a specialist clinic with apparently refractory epilepsy did not have epilepsy at all (Smith et al., 1999). The studies note the importance of distinguishing between nonspecific EEG abnormalities (such as nonfocal slowing) and specific EEG changes (spikes, sharp waves, spike-wave, and sharp/slow wave). The latter are clearly much more common in people with epilepsy, although a study in air cadets suggests that they can occur in 0.5-2% of healthy individuals (Gregory et al., 1993; Reuber et al., 2002b).

Plainly, if a spontaneous epileptic seizure is recorded on vEEG, then the diagnosis of epilepsy is established. Unfortunately the negative predictive value of the absence of epileptiform activity during a 5-day vEEG monitoring period for the diagnosis of epilepsy is unknown.

The only hard evidence that a patient does not have epilepsy is provided by an absence of epileptic seizures, off antiepileptic drugs (AEDs). Some patients with epilepsy may have a low untreated seizure frequency, so a long period of clinical monitoring may be needed. In the great majority of patients with PNES, it should be possible to be sufficiently certain about the absence of additional epileptic seizures on the basis of history and simple investigations (such as interictal EEG recordings and brain MRI) to allow clinicians to withdraw erroneously started AEDs. It has been shown that the risk of "unmasking" epileptic seizures by withdrawing AEDs is very low if a patient with PNES conforms to a small number of simple criteria (Oto et al., 2005). Where the risk of or clinical concern for epilepsy *is* higher, but a definitive diagnosis of epilepsy cannot be established by other means, withdrawal of AED can be undertaken under inpatient observation (Duncan, 2010), followed by further monitoring on an outpatient basis.

Increasingly, with earlier recognition, patients with PNES present before being started on AEDs. Few such patients have PNES and additional epilepsy (Duncan et al., 2011), and the prevalence of epileptiform EEG abnormality will be low enough to make false positives a concern. In these patients, the exclusion of epilepsy depends principally on clinical monitoring.

Some have alluded to a post hoc confirmation of PNES by seeing a resolution of symptoms after communicating the diagnosis. Remission of events after communication of the diagnosis, or after psychological intervention, occurs in a subset of patients with PNES (Farias et al., 2003; Hall-Patch et al., 2010). In up to 30% of patients (McKenzie et al., 2010), possibly more when events are of recent onset (Duncan et al., 2011), PNES cease immediately after the patient has been provided with an explanation of their condition. A significantly large proportion of patients relapse, however, if appropriate treatment is not provided (Wilder et al., 2004). Nonetheless, such a response could be documented when the diagnosis of PNES is being considered and could augment other diagnostic data (bearing in mind that some epilepsies also take a variable course with periods of higher and lower seizure frequency).

SUMMARY

This report provides a summary of current diagnostic approaches used for the diagnosis of PNES. The gold standard for PNES diagnosis is vEEG monitoring, where the ictus is observed, simultaneously co-registered with EEG. When a patient's habitual event is captured on vEEG, and vEEG is used with patient history, there is excellent IRR, and the diagnosis of PNES is made with high confidence. Both the observation of an event alone and the history alone, are subject to false positives and false negatives, which lead to misclassification/misdiagnosis of epileptic seizures as PNES, or PNES as epilepsy. Other tests are commonly used to aid in differentiation of ES from PNES, including PRL and neuropsychological batteries. The lack of specificity and sensitivity renders these and other tests noted above as adjuncts, which do not replace vEEG.

In the absence of vEEG in many practices and in the developing world, this report provides information on the

diagnostic value of ancillary tests and a categorization of different levels of diagnostic certainty, which can be used to communicate clinical or research data. Clinically, even a diagnosis of "possible PNES" may prompt a discussion of psychological evaluation in some cases (for instance, when more a definitive diagnosis cannot be made, and a patient may be willing to engage in a comprehensive evaluation for seizures). Although some research studies may include only patients with definite diagnoses, that is, vEEG confirmed "documented PNES," other studies may be less exclusive depending on resources available or the nature of the research question. Different studies can be more readily comparable in the future if authors provide a breakdown of the proportion of patients in different certainty categories. This classification approach is not without precedent. Similar diagnostic categories of possible, probable, clinically established and documented/definite are used in research and clinical practice for the dementias (McKhann et al., 1984) and for other psychogenic disorders, including psychogenic movement disorders (PMD) (Fahn & Williams, 1988). A limitation of this consensus report is that the proposed categories have not been assessed with a sensitivity/specificity analysis. Future research for the proposed categories in this consensus statement could be assessed, as has been done for dementias (Loewenstein et al., 2001) and PMD diagnostic criteria (Shill & Gerber, 2006), to establish sensitivity and specificity.

There is also a need for future research to focus on the following:

- 1 Reducing the delay in diagnosis of PNES.
- **2** Improving the transition from neurologic to mental health treatment for PNES.
- **3** Identifying predictors of treatment resistance and treatment response.
- 4 Multicentered, fully powered, randomized controlled trials for pharmacologic and psychotherapeutic management of patients with PNES and their families.

In conclusion, the ability to diagnose PNES when vEEG is not available may open opportunities to lower and middle income countries where monitoring is not available.

DISCLOSURE

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Table S1. Neuropsychological measures in PNES.